(PRA and PRC), renal and cardiac hypertrophy in NALP3 and ASC deficient mice on which the two- kidney, one clip (2K1C) and the deoxy corticosterone-acetate (DOCA)/salt models were applied. MBP, PRA, PRC, and HW/BW were significantly increased at week 12 in WT-2K1C mice compared to the sham. Neither NALP3-KO nor ASC-KO 2K1C treated mice developed hypertension and had lower circulating levels of PRA and PRC but serum amyloid A (SAA) and IL6 compared to the control. RNA levels of SAA, NLRP3, IL1β and IL1α were increased in the ischemic kidney in C57BL/6J mice. Administration of anti-IL1β antibody to the WT mice attenuated the increases of blood pressure and renin in 2K1C mice. With chronic administration of DOCA/salt, MBP in both NLRP3-KO and WT mice were comparable and not significantly increased compared to tap water group. PRA and PRC in both NALP3-KO and WT mice were significantly suppressed by DOCA/salt. HW/BW and KW/BW in both DOCA/salt treated NALP3-KO and WT mice were significantly increased. DOCA/salt induced hypokalemia was comparable between Nlrp3 KO and WT groups. However, the heart and kidney index in DOCA/salt NLRP3-KO mice was significantly lower than that in DOCA/salt WT mice. Our data show that NLRP3 mediated IL1β is linked to development of renovascular hypertension and suggest that a novel target for the treatment of hypertension. The results also implicate that NLRP3 contributes to the development of cardiac and renal hypertrophy independently of blood pressure in the DOCA/salt model.

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263 THE IMPACT OF BODY WEIGHT MANAGEMENT IN CHRONIC KIDNEY DISEASE PATIENTS WITH OBESITY
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Chronic kidney disease (CKD) and obesity are important public health concerns. We examined the influence of dietary management and physical exercises on renal function, lipid profiles and exercise tolerance of a group of CKD patients. We enrolled 45 obese patients with CKD from stage 1 to 3b with tailored by regular dietitian consultation, recommendations of adequate daily protein and caloric intake, and physical exercises for body weight (BW) reduction. The target was BW reduction ≥ 5%. Thirty eight obese CKD patients completed the study and 24 patients reached the target. Patients who reached the target was significant larger in total body fat decrement than those who failed (3.0% vs 0.5%, p = 0.003). In addition, total muscle mass, especially on trunk and lower limbs (1.2% vs 0%, p = 0.026) was significant decreased in patients who reached the target compared to those who failed. The laboratory data disclosed the significant reduction of Chol (0.7% vs. -19.3%; p = 0.007), LDL-C (-3.7% vs. -37.6%; p < 0.001), and UA (0% vs. -0.8%; p = 0.025) between the two groups. The cardiorespiratory endurance to complete 800 meters run (375.1 ± 64.7 sec vs. 327.1 ± 84.0 sec; p = 0.001), abdominal muscle strength and endurance by times of sit-ups (13.6 ± 9.1 number/min vs. 19.3 ± 9.2; p = 0.005), and flexibility of the lower back by sit and reach (18.8 ± 10.8 cm vs. 27.8 ± 10.9 cm; p = 0.001) of the patients who reached the target were improved significantly after 2-months of physical exercise training and education. We conclude that combination of dietary management and exercises are effective in improving health-related physical fitness, blood pressure control, dyslipidemia and renal function in overweight or obese CKD patients. Supportive individualized programs for lifestyle change could exert beneficial effects. A longitudinal study with larger sample size is warranted to elucidate the efficacy of combination of dietary management and exercises.

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264 CXL16 REGULATES ANGIOTENSIN II-INDUCED INFLAMMATION AND PROTEINURIA
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Hypertension is a major cause of chronic kidney disease. Recent studies have shown that inflammation plays a critical role in the pathogenesis and progression of hypertensive kidney disease. However, the signaling mechanisms underlying the induction of inflammation are poorly understood. We have found that CXL16, a recently discovered chemokine, is induced in the kidneys in a mutant model of angiotensin II (Ang II)-induced hypertension. Therefore, we examined whether CXL16 regulates inflammatory cell infiltration and renal injury using CXL16 knockout (CXL16-KO) mice. Wild-type (WT) and CXL16-KO mice were treated with Ang II via subcutaneous osmotic minipumps at 1500 ng/kg/min for up to 4 weeks. To accelerate renal injury, all mice were subjected to unilateral nephrectomy and received 1% NaCl in drinking water. WT and CXL16-KO mice had virtually identical blood pressure at baseline. Ang II treatment led to an increase in blood pressure that is similar between WT and CXL16-KO mice. Immunohistochemical analysis showed that targeted disruption of CXL16 inhibited Ang II-induced inflammation. In conclusion, our results indicate that CXL16 plays a pivotal role in the development of renal injury and proteinuria through regulation of macrophage and T cell infiltration.

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265 STUDY MECHANISMS OF LOW PROTEIN DIET SUPPLEMENTED WITH KETOANLOGS ON REDUCING PROTEINURIA AND MINTINING NUTRITIONAL STATUS IN TYPE2 DIABETIC NEPHROPATHY
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Diabetic nephropathy is one of the most important causes of end stage renal disease (ESRD) in the world. Recently, randomized controlled clinical trial performed in China, it was found that compound 3-keto acid tablet in combination with low protein diet(LPD + KA) reduced proteinuria and delayed the progression to ESRD Type 2 diabetic nephropathy(T2DN). However, the mechanisms underlined these effects were not been elucidated. This study is a part of the continuation of the study to explore the effects of LPD + KA on podocyte as well as local RAS in the kidney. A total of 60 patients with T2DN and CKD stages 3-4 are included in this study. All patients are treated with a LPD containing 0.6g protein/kg BW per day and 120-125 kg/kg BW per day. The diet is randomly supplemented with keto-amino acids (Ketosteril®, Fresenius Kabi) at a dosage of 100 mg/kg BW per day. The other 30 patients receive placebo. After six months of follow-up, protein intake differed only by 0.07 g/kg/day between the two groups. The mean declines in GFR were 4.2 mL/min/1.73 m² (95%CI; 2.9 to 5.5) in LPD group and 3.7 (95%CI; 2.7 to 4.7) in LPD + KA group. Compared with LPD, patients in LPD + KA group had reduced proteinuria (2.16 ± 0.4 vs 3.56 ± 0.6 g/day; p < 0.05). However, serum albumin and prealbumin levels did not change. Through further research we found that, after a median follow-up for six months, the urinary angiotensinogen:creatinine ratio in LPD patients was higher than in LPD + KA subjects (10.1 ± 3.2 μg/mg vs 5.8 ± 1.2 μg/l; p < 0.05).The urinary mRNA levels of the target genes nephrin, podocin and synaptopodin of podocyte were higher in the LPD group compared with LPD + KA (p = 0.05) in renal sediment, and the expression of nephrin, podocin and synaptopodin mRNA positively correlated with serum creatinine (r = 0.326, p = 0.04; r = 0.426, p = 0.03; r = 0.343, p < 0.001, respectively). In conclusion, compound 3-keto acid tablet in combination with low protein diet (LPD + KA) is associated with amelioration of proteinuria, better reduction in the loss of kidney function compared with LPD alone meanwhile nutrition status remained well. The mechanism under these effects may be explained by the role of LPD + KA diet in reducing urine podocyte loss and lowering the angiotensinogen level in the urine.

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266 CLINICAL STUDY OF THE EFFECTS OF LOW-PROTEIN DIET AND SUPPLEMENTED WITH 3-KETOACIDS THERAPY ON NUTRITION STATUS AND RESIDUAL RENAL FUNCTION IN CONTINUOUS AMBULATORY PERITONEAL DIALYSIS(CAPD) PATIENTS
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It is critical to preserve residual renal function (RRF) in CAPD, as RRF is associated with lower morbidity and mortality. low- protein diet supplemented with 3-keto acids was reported to have an important roles in delaying in follow-up period progression of renal failure and improving nutritional status in non-dialysis patients with chronic renal failure. We evaluate the effects on the nutritional status and RRF of a low-protein diet supplemented with 3-keto acids on CAPD patients prospectively. Seventy eight CAPD patients who were...